NHANES II

(21)

NATIONAL ESTIMATES OF BLOOD LEAD LEVELS: UNITED STATES, 1976–1980

Association with Selected Demographic and Socioeconomic Factors

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Abstract Data from the second National Health and Nutrition Examination Survey showed that 22 per cent of persons six months through 74 years old had blood lead levels under 10 μg per deciliter; 1.9 per cent had elevated levels (≥30 μg per deciliter [≥1.45 μmol per liter]). Among children six months through five years old the prevalence of elevated levels was significantly higher (4 per cent) than previously predicted on the basis of fewer data. The prevalence of elevated lead levels was 12.2 per cent in black children and 2.0 per cent in white children. Mean levels of blood lead were higher in blacks than whites among children and adults, among young children living in urban and rural areas, and among members of low-income, moderate-income, and higher-income families. These racial contrasts may reflect different lead exposure or absorption (or both). Young children from families (both white and black) whose incomes were under $6,000 had a significantly higher prevalence of elevated lead levels than those from households with incomes of $6,000 or more. (N Engl J Med. 1982; 307:573-9.)

UNDUE exposure to lead and lead toxicity, particularly among young children, have remained public-health problems for decades.[1,2] During the 1970s several revisions were made in designating the level of blood lead deemed important in programs designed for the screening, diagnosis, treatment, and follow-up of children with undue lead absorption and lead poisoning.[3-5] These changes in diagnostic criteria reflected an increased awareness of the extent to which health can be affected by lead exposure. Previous estimates of the prevalence of undue lead absorption among high-risk pediatric age groups ranged from greater than 40 per cent in some cities[6] during the mid-1960s to approximately 5 per cent as reported to the Centers for Disease Control during the first six months of fiscal year 1981 by participating community programs for the prevention of childhood lead poisoning.[7] Because of interest in the general population, blood lead concentrations were determined in a nationally representative sample of persons examined in the second National Health and Nutrition Examination Survey (NHANES II). The purposes of our report are to present descriptive data on blood lead levels in the United States population as a whole and to determine the association of blood lead levels in the population (including concentrations over 30 μg per deciliter [1.45 μmol per liter]) with specific socioeconomic and demographic variables, including annual family income and degree of urbanization of the place of residence.

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METHODS

The NHANES II Sample Design

The NHANES II, conducted between 1976 and 1980, used a multistage probability design that involved selection of primary sampling units, segments (clusters of households) within these units, households, eligible persons, and finally sample persons. Primary sampling units typically were composed of a county or group of contiguous counties. A detailed description of the survey design has been published.[8] A total of 27,801 persons from 64 sampling areas were selected in the probability sample as representative of the United States civilian population six months through 74 years old who were not institutionalized. Certain subgroups in the population that were of special interest for nutritional assessment were oversampled: preschool children (six months through five years old), persons 60 through 74 years old, and the poor (persons living in areas defined as poor by the United States Bureau of the Census for the 1970 census).[8] The United States Bureau of the Census selected the NHANES II sample according to rigorous specifications from the National Center for Health Statistics so that the probability of selection for each person in the sample could be determined.

The statistics presented in this report are population estimates. The laboratory findings for each person in the sample have been inflated by the reciprocal of selection probabilities, adjusted to account for persons who were not examined, and stratified afterward according to race, sex, and age, so that the final weighted population estimates closely approximated the civilian noninstitutionalized population of the United States as estimated independently by the United States Bureau of the Census at the midpoint of the survey, March 1, 1978.

Demographic and Socioeconomic Terms

Age was defined as the subject's age at the time of the interview. The medical-examination phase of the study was scheduled from

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one to four weeks after demographic and medical-history information were collected through an interview in the household.

Race was recorded as white, black, or "other." The last category included American Indians, Chinese, Japanese, and all other races not white or black. Mexicans were included with whites unless they were definitely known to be American Indian or of another race. Persons of mixed black and other parentage were recorded as black. Selection of the racial category of a subject was made by the interviewer.

Annual family income was the total income received during the 12 months before the interview by related persons living in the household. Respondents were asked to include income from all sources, such as wages, salaries, Social Security or retirement benefits, financial help from relatives, rent from property, and similar sources.

Urbanization status was the degree of population density of the place of residence, according to the definitions of "urban" and "rural" for the 1970 census.[9] The categories used in this report were urbanized areas with 1 million or more persons, urban areas with fewer than 1 million persons, and rural areas. The second category included urbanized areas and small urban areas of more than 2500 persons. Urbanized areas with 1 million or more inhabitants were divided into "central cities" and "non-central cities" within the census definition of a standard metropolitan statistical area.

Blood Lead Determination

Lead concentrations in whole-blood specimens and control samples from the NHANES II were determined by a modified microcup atomic-absorption method.[10] Specimens were analyzed in duplicate; the average of the two analyses was used for the statistical analysis.[11] Two quality-control systems were used: "Bench" quality-control samples were inserted by the analyst and measured in duplicate in each analytic run to allow the analyst to make judgments on the day of analysis, and "blind" quality-control samples were placed in vials, labeled with false patient-identification numbers, and processed so that they were indistinguishable from regular NHANES II samples. Details of the quality-control systems have been previously reported.[11,12] At least one blind sample was randomly incorporated with every 20 NHANES II samples and analyzed in duplicate.

The standard deviation for the "normal blind" pool, with a mean of 13.7 μg per deciliter (0.66 μmol per liter), was 2.2 μg per deciliter (0.11 μmol per liter), and that for the "elevated blind" pool, with a mean of 25.5 μg per deciliter (1.23 μmol per liter), was 3.2 μg per deciliter (0.15 μmol per liter). A blind pool with a concentration of 30 μg per deciliter (1.45 μmol per liter) or below was used; since all blood lead values above 30 μg per deciliter from persons tested during the NHANES II were telephoned to their physicians for follow-up. The coefficients of variation for the bench quality-control samples with lead levels of 30 μg per deciliter (1.45 μmol per liter) or above ranged from 7 to 15 per cent.[12]

Statistical Analysis

The statistical methods used to analyze the data took into account the complex survey design of the NHANES II.[9] The standard errors of the weighted means and the proportions of persons with elevated blood lead levels were calculated with the Taylor series-linearization method.[13] In the analysis of blood lead levels, the population was divided into three age groups: children six months through five years old, youths six through 17 years old, and adults 18 through 74 years old. Regression analysis was performed within each age group; the blood lead concentration was used as the dependent variable, and age was used as a covariate. The effect of each of the demographic variables (race, sex, income, and degree of urbanization) on the blood lead concentration was tested in this analysis after adjustment for age.

Tests of the hypothesis of no difference in the proportions of undue lead absorption among different subgroups in the population were performed with the Grizzle–Starmer–Koch approach to categorical data analysis.[14] This analysis involved two stages: estimation of the proportions with undue lead absorption for subgroups of interest, and estimation of an appropriate variance-covariance matrix and hypothesis testing using categorical data analysis. The computing for this analysis involved a combination of two computer programs[15,16]: SURREGR for the first stage of calculations and GENCAT, a program for generalized chi-square analysis of categorical data, for the second stage. A detailed discussion of this method has been published by the National Center for Health Statistics.[17]

Limitations of the Data

Although rigorous quality-control methods were implemented throughout specimen collection and processing and in data processing to ensure the validity and accuracy of the results reported, the reader should be aware of some factors that may have had effects on the data. The first is the degree of imprecision of blood lead measurements. On the basis of analyses of the quality-control pools, the coefficients of variation (i.e., the standard deviations expressed as percentages of the mean blood lead levels for a given pool) were on the average 12.0 per cent and 15.0 per cent for pool mean values above and below 30 μg per deciliter (1.45 μmol per liter), respectively.[12]

Of the 27,801 persons, 16,563, including all children six months through six years old and a half sample of persons seven through 74 years old, were asked to provide blood samples for blood lead measurements. Approximately 39 per cent of these sample persons had missing lead values because of nonresponse at various stages of the survey. The percentage of nonresponse was comparable when the subjects' race, sex, annual family income, or degree of urbanization of residence was considered.[17] However, the rate of nonresponse was age-dependent. Over half the children six months through five years old (51 per cent), as compared with 28.6 per cent of youths six through 17 and 35.7 per cent of adults 18 through 74, had no blood lead determinations.

The national estimates presented in the results are based on data obtained in 9933 NHANES II subjects whose blood lead levels ranged from 2.0 to 66.0 μg per deciliter (0.096 to 3.18 μmol per liter) and who received venipuncture. The potential for contamination during the finger-stick collection process is recognized.[18] Statistical analysis of the unweighted data[11] suggested that inclusion of the data from finger sticks in this analysis would have introduced bias to the estimates of mean levels in children. Overall, among children six months through five years old, the unweighted mean blood lead level in those receiving finger sticks (108 subjects) was observed to be approximately 6.0 μg per deciliter (0.29 μmol per liter) higher than that in children receiving venipunctures. This observed mean difference was consistent in blacks and whites. In addition, three subjects who received venipunctures had blood lead concentrations of >70.0 μg per deciliter (>3.38 μmol per liter). These were extreme cases of lead exposure, and the levels were unusually high in the light of the remaining distribution of blood lead levels in the general population. Because of the potential effects on the variances of national estimates, the values obtained by finger stick and those in three extreme cases were excluded from further stages of our analysis.[17]

A possible logistic factor indirectly influencing the blood lead data was the itineraries of the mobile examination centers. In order to minimize the effects of adverse weather conditions on survey operations, mobile examination centers were set up in the northernmost states during the summer and in more southern states during the winter. The potential effect of seasonality[19] on blood lead levels is one aspect of the association (or lack of association) between blood lead levels and selected demographic factors, specifically regional differences in blood lead concentrations.

Results

Association with Age, Race, and Sex

The mean, median, and S.E.M. of blood lead concentrations by age in persons six months through 74 years old are presented in Table 1. Mean blood lead levels by age and race and by age and sex are shown in Figures 1 and 2, respectively. To evaluate the associations of age, race, and sex with blood lead levels, a statistical analysis was performed (as in previously described programs[15,16]) within three age groups: young children six months through

five years old, youths six through 17, and adults 18 through 74.

For children under six, there was no statistically significant association ($\alpha = 0.05$) between age and mean blood lead level. For children and youths six through 17, there was a statistically significant relation between age and mean blood lead concentration ($P<0.001$). In general, mean blood lead values declined with increasing age until adolescence (15 through 17 years). Among adults 18 through 74, there was also a significant trend in blood lead level with age ($P<0.001$): Mean blood lead levels were positively associated with age until the middle ages (45 through 54 years), with a moderate decline in the older age groups.

Race and mean blood lead concentrations were significantly associated within each of the three age groups used for analysis ($P<0.001$). Data by age for races other than white and black were not reported, because further subcategorization resulted in group sizes judged too small to be reliable estimators for the general population.

As shown in Figure 1, mean blood lead levels were consistently higher in blacks than in whites across all ages. Overall, among children six months through five years old, blood lead levels in blacks were on the average 6 μg per deciliter (0.29 μmol per liter) higher than those in whites (Table 2). In addition, the prevalence of blood lead concentrations of ≥30 μg per deciliter (≥1.45 μmol per liter) was much higher among black preschool children than among white preschool children (Table 2).

Among young children, the sex of the subject was not significantly associated with the mean blood lead concentration. Among youths six through 17, the difference in mean blood lead level between boys and girls increased progressively with age. For adults 18 through 74, mean blood lead concentrations were consistently and significantly higher in men than in women across all age groupings ($P<0.001$) (Fig. 2).

Table 1. Blood Lead Levels by Age in All Races in the United States, 1976–1980.*

Age Group	Estimated Population	No. of Persons Examined †	Blood Lead Level ‡	Median Blood Lead Level
yr	thousands		μg/dl	
0.5–2	7,676	759	16.3±0.57	15.0
3–5	9,186	1613	15.9±0.40	15.0
6–8	10,259	451	13.9±0.47	13.0
9–11	10,621	377	12.9±0.39	12.0
12–14	11,632	448	11.4±0.32	11.0
15–17	12,452	444	12.1±0.35	11.0
18–24	27,448	985	13.1±0.33	12.0
25–34	32,752	1041	13.7±0.33	13.0
35–44	23,651	753	14.6±0.36	13.0
45–54	23,032	724	15.3±0.32	14.0
55–64	20,350	1149	14.6±0.32	14.0
65–74	14,496	1189	14.4±0.23	13.0
Total	203,554	9933	13.9±0.24	13.0

*At the midpoint of the second National Health and Nutrition Examination Survey, March 1, 1978.

†Subjects with venous-blood samples.

‡Means ±S.E.M. To convert blood lead values to micromoles per liter, multiply by 0.04826.

Figure 1. Blood Lead Levels by Race and Age in the United States According to the Second National Health and Nutrition Examination Survey, 1976–1980.

To convert blood lead values to micromoles per liter, multiply by 0.04826.

Associations with Income and Degree of Urbanization

The associations of the family income and the degree of urbanization with the blood lead level were generally consistent across all three broad age groups in the population. However, they were most pronounced in children six months through five years old. Hence, further considerations of blood lead levels are limited to preschool children. Attempts to include more cross-classifications of these variables resulted in group sizes judged too small to be reliable estimators for the general population. For example, although it would have been of interest to determine whether the association between race and blood lead level differed between various degrees of urbanization by income groups, the number of subjects within such subgroups was too small.

As the family income increased, the mean blood lead concentration in young children decreased (Table 3). Differences between blacks and whites in mean blood lead values at all three levels of family income were significant ($P<0.01$). There was a significantly higher prevalence of persons with blood lead levels of ≥30 μg per deciliter (≥1.45 μmol per liter) among black preschool children from low-income families than among other preschool children from the same or other income groups ($P<0.01$).

Across the three categories of urbanization, the mean blood lead level in young children increased with the degree of urbanization of the areas where they lived (Table 3). When differences between urban and rural groups were examined separately for black and white children, it was again observed that blacks had significantly higher mean blood lead concentrations than those of whites in large urban, smaller urban, and

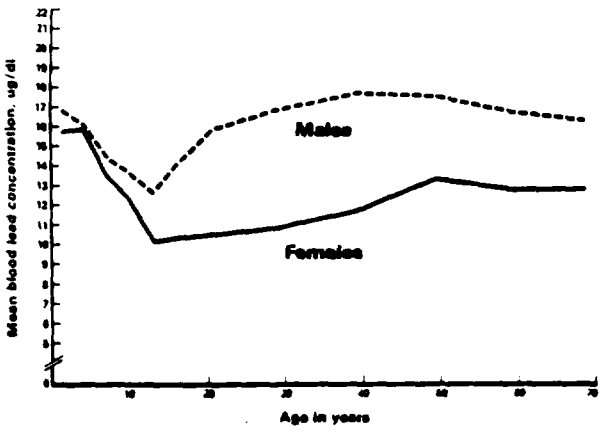

Figure 2. Blood Lead Levels by Sex and Age in the United States According to the Second National Health and Nutrition Examination Survey, 1976–1980.

To convert blood lead values to micromoles per liter, multiply by 0.04826.

rural areas ($P<0.01$). Since a large proportion of urban black children live in the central cities, it might be expected that the higher blood lead values among blacks would reflect differences in the degree of urbanization of their place of residence. However, the relatively higher mean blood lead levels in all three urban or rural groups demonstrated that the observed racial effects were not simply a reflection of urbanization status. Further investigation of blood lead levels in large urban areas revealed that the mean values in black children living in the central cities were 3.9 μg per deciliter (0.19 μmol per liter) and 4.8 μg per deciliter (0.23 μmol per liter) higher than those in black children living in non-central cities and rural areas, respectively (Table 3). These differences were not significant. However, within the central cities, the mean blood lead levels in black children were significantly higher than those in white children ($P<0.01$).

Prevalence of Elevated Blood Lead Levels among Young Children

The consistent racial differences in blood lead concentrations among children six months through five years old and the presence of higher blood lead concentrations among those in the low-income groups and large urban areas were also found with regard to the percentage of children with blood lead levels of 30 μg per deciliter (1.45 μmol per liter) or more.

Overall, 12.2 per cent of blacks, as compared with 2.0 per cent of whites, had blood lead values of 30 μg per deciliter (1.45 μmol per liter) or more. This difference was significant for both boys and girls ($P<0.01$). The percentage of elevated blood lead levels was slightly higher among boys than girls, but this difference was not significant. There was a significant association between income and race ($P<0.01$), with a stronger inverse relation among blacks than among whites between income and the proportion of children with elevated blood lead levels (Table 3). According to the Center for Disease Control's guidelines for elevated blood lead levels (≥30 μg per deciliter [≥1.45 μmol per liter]), it is estimated from the NHANES II data that almost one fifth (18.5 per cent) of black children from low-income families — the group with the highest proportion of elevated blood lead levels — should be referred for medical follow-up. Among both whites and blacks, the percentage of children with elevated blood lead levels was lowest in the highest income group. There was also a significant interaction between the degree of urbanization and race ($P<0.01$) (Table 3). The relation between the percentage of children with elevated blood lead levels and the degree of urbanization was apparently stronger in blacks than in whites. In the central cities, the percentage of children with elevated blood lead levels was significantly higher among blacks than among whites ($P<0.01$). Even in the smaller urban and rural areas, 10.2 per cent of black children had elevated blood lead levels, as compared with fewer than 2.0 per cent among whites. Caution should be exercised in the interpretation of racial differences in rural areas because of the relatively small number of rural black children examined (42 cases).

Discussion

Blood Lead Levels in the Population

Data on blood lead concentrations collected in the NHANES II provide the first population estimates

Table 2. Blood Lead Levels by Race and Age in the United States, 1976–1980

Race and Age	Estimated Population	No. of Persons Examined †	Mean Blood Lead Level ‡	Median Blood Lead Level	Blood Lead Level (μg/dl)						
					0	10–19	20–29	30–39	40–49	50–59	60–69
	(thousands)*		μg/dl		% distribution						
White											
6 mo–2 yr	6186	589	15.0±0.56	14.0	16.1	64.1	17.3	2.2	0.0	0.2	0.0
3–5 yr	7455	1287	14.9±0.41	14.0	13.6	69.1	15.4	1.6	0.1	0.0	0.0
Black											
6 mo–2 yr	1164	141	20.9±0.96	19.0	0.3	50.2	34.2	13.0	1.4	0.5	0.4
3–5 yr	1421	278	20.8±0.55	20.0	3.8	42.5	43.3	8.5	1.4	0.5	0.0

*Estimated population at the midpoint of the second National Health and Nutrition Examination Survey, March 1, 1978.

†With lead determinations from blood specimens obtained by venipuncture.

‡Means ±S.E.M. To convert blood lead values to micromoles per liter, multiply by 0.04826.

Table 3. Blood Lead Levels in Children Six Months through Five Years Old by Annual Family Income and Degree of Urbanization of Place of Residence in the United States, 1976–1980.

Demographic Variable	All Races *			White			Black			Prevalence of Blood Lead Levels > 30 μg/dl ‡		
	Estimated Population †	No. of Persons Examined	Blood Lead ‡	Estimated Population †	No. of Persons Examined	Blood Lead ‡	Estimated Population †	No. of Persons Examined	Blood Lead ‡	All Races *	White	Black ¶
	thousands *		*μg/dl*	*thousands* *		*μg/dl*	*thousands*		*μg/dl*	*% of persons examined*		
Annual family income ¶												
<$6,000	2465	448	20.0±0.6	1408	256	18.1±0.6	917	176	22.9±0.9	10.9±1.4	5.9±1.3	18.5±3.6
$6,000–14,999	7534	1083	16.2±0.5	6252	887	15.3±0.5	1037	163	20.7±0.6	4.2±0.7	2.2±0.5	12.1±1.9
≥$15,000	6428	774	14.1±0.4	5707	690	13.7±0.4	502	60	17.2±0.8	1.2±0.4	0.7±0.3	2.8±1.2
Degree of urbanization												
Urban ≥1,000,000 persons ‖	4344	544	18.0±0.5	3112	358	16.6±0.6	1093	172	22.2±0.8	7.2±0.7	4.0±0.7	15.2±1.5
Central cities	1822	286	20.0±0.7	885	133	17.4±0.8	855	143	23.1±1.3	11.6±1.9	4.5±1.9	18.6±2.8
Non-central cities	2519	257	16.5±0.6	2223	224	16.2±0.6	238	29	19.2±0.7	3.7±0.8	3.8±0.8	3.3±1.4 **
Urban <1,000,000 persons	6891	944	16.5±0.7	5297	699	15.4±0.7	1246	205	20.3±0.8	3.5±0.6	1.6±0.4	10.2±2.4
Rural	5627	884	13.9±0.6	5233	819	13.5±0.6	245	42	18.3±2.6	2.1±0.9	1.2±0.5	10.3±5.3 **

*Includes data for races not shown separately.

†Estimated at the midpoint of the second National Health and Nutrition Examination Survey, March 1, 1978.

‡Means ±S.E.M. To convert blood lead values to micromoles per liter, multiply by 0.04826.

§One child (a black boy with a family income under $6,000, living in a rural area, with a blood lead level of 76 μg per deciliter) was excluded. This exclusion had a negligible effect on the national estimates shown here.

¶All values shown for this variable reflect the exclusion (from analyses and tests for significance) of children in households that declined to report their income.

‖A child not specified as living in either a central or a non-central city was included in the calculation of values shown for this entry, but was excluded from the calculation of values shown for central and non-central cities.

**Fewer than 50 persons in the sample cell.

that are descriptive for the United States. Because of the nature of the sample design of the NHANES II, relatively few other studies are appropriate for comparison. In interpreting associations between blood lead levels and the demographic variables identified in this report, it is essential to recognize that the NHANES II did not include estimates of environmental lead exposure. Accordingly, differences in blood lead levels observed between population groups may reflect different degrees of lead exposure, variation in lead absorption or in the metabolic response to lead, or a combination of these factors. Blood lead levels have been reported in groups of people who were of interest because their lead exposures were unusually high (e.g, children living near metal smelters[20,21]) or unusually low (e.g., inhabitants of nonindustrialized, remote regions[22,23]). NHANES II data show that a wide range of blood lead levels occurs within the general United States population. Twenty-two per cent of the population had blood lead concentrations of <10 μg per deciliter (<0.48 μmol per liter), whereas 1.9 per cent across all age groups had levels of ≥30 μg per deciliter (≥1.45 μmol per liter). There were highly significant differences in blood lead levels in specific subpopulations. During the past 50 to 100 years, the majority of nonindustrial lead toxicity has occurred among children.[1,2,24] The same pattern is observed — i.e., NHANES II data indicate that children under six years old had higher mean blood lead levels than those of children from six until approximately 15 through 17 years. Through the 1960s, pediatric lead toxicity was regarded as largely an urban health problem, more or less localized to deteriorating areas of central cities (among many others, see Griggs et al.[25]). Blood lead levels observed among preschool children living in these areas were reported to average 40 to 50 μg per deciliter (1.93 to 2.41 μmol per liter).[6] In the NHANES II, blood lead levels in black preschool children living in urban areas with 1 million or more inhabitants averaged 22.9 μg per deciliter (1.10 μmol per liter). Among whites from a comparable subpopulation, the mean blood lead concentration was 18.1 μg per deciliter (0.87 μmol per liter).

Specific rural populations were recognized as being at risk for excessive exposure to lead. Under circumstances involving an important source of lead emissions, such as a smelter, the observed blood lead concentrations can be greatly elevated. Landrigan et al.[21] found that 55 per cent of one to four-year-old children living within 1.6 km of a smelter in Kellogg, Idaho, had blood lead concentrations of 40 to 59 μg per deciliter (1.93 to 2.85 μmol per liter). Even if such severe lead contamination is not present, specific subgroups of rural populations are at greater risk for elevated blood lead levels. For example, Perrin and Merkens reported that the prevalence of blood lead concentrations of ≥30 μg per deciliter (≥1.45 μmol per liter) was approximately 2.5 times higher in 12-month-old to five-year-old children of migrant farm workers than in children of nonmigrant workers from an economically similar group living in the same rural area.[26] NHANES II data indicated that in the general population, blood lead values were highest among urban dwellers, especially those living in central cities, and became progressively lower as the degree of urbanization declined.

Perhaps the most striking observation from the NHANES II data was that blood lead levels were consistently higher among blacks than among whites; this difference was found in children and adults, in rural residents and urban dwellers, and in families with low, moderate, and high incomes. No clear-cut reason for the consistently higher mean blood lead concentrations observed among black children can be concluded from the results of this study; however, these results support the findings of other studies with regard to this racial difference. In a report summarizing data obtained in New York City programs for the prevention of childhood lead poisoning between 1970 and 1976, Billick et al. observed that among preschool children blacks had higher blood lead levels than whites.[27] Other reports of higher blood lead levels among blacks have been published[28,29]; however, the groups contrasted were different geographically and possibly economically, so that differences in blood lead levels could not be attributed to race alone.

In this study and others,[30-33] men have been found to have higher blood lead concentrations than those of women. Some of this difference appears to have been associated with a higher potential for occupational exposure of men to lead. This sex-related difference was similar among white and black persons.

Concerns over the Prevalence of Elevated Blood Lead Levels

In estimating the prevalence of elevated blood lead levels in the pediatric population from NHANES II data, the criterion of 30 μg per deciliter (1.45 μmol per liter) of whole blood, established by the Centers for Disease Control in 1978,[5] has been used. If this concentration occurs in combination with an erythrocyte protoporphyrin concentration of 50 to 250 μg per deciliter (0.9 to 4.4 μmol per liter) of whole blood, the child is thought to have undue lead absorption. Lead poisoning was defined by the Centers for Disease Control[5] with particular combinations of blood lead concentrations and degrees of elevation of the erythrocyte protoporphyrin level. However, lead poisoning was defined by blood lead alone if a whole-blood lead concentration of ≥70 μg per deciliter (≥3.38 μmol per liter) was confirmed. Community-based lead-poisoning-prevention programs, analyzing venous-blood samples for both erythrocyte protoporphyrin and lead, report that approximately 75 per cent of children with blood lead levels of ≥30 μg per deciliter (≥1.45 μmol per liter) also have erythrocyte protoporphyrin values of ≥50 μg per deciliter (≥0.9 μmol per liter) (Houk V: unpublished data). Erythrocyte protoporphyrin levels were measured in subjects of the NHANES II, but these data were not available at the time of this report. Although very few persons tested in the NHANES II had blood lead levels in excess of 70 μg per deciliter (3.38 μmol per liter), an estimated 4 per cent of United States children six months through five years old (approximately 675,000) have elevated blood lead levels (≥30 μg per deciliter and <70 μg per deciliter [≥1.45 μmol per liter and <3.38 μmol per liter]). The same groups who have higher mean blood lead levels also have a higher prevalence of elevated blood levels: preschool children from low-income families living in highly urbanized areas, especially central cities, with the prevalence highest among blacks. Overall, blacks six months through five years old had a sixfold higher prevalence of elevated blood lead levels than whites (3.8 times greater in highly urbanized areas and 7.3 times higher in smaller urban and rural areas). Among whites and blacks, preschool children from families with annual incomes under $6,000 had a significantly higher prevalence of elevated blood lead levels than that of children from families whose incomes were over $6,000 (3.9 times greater for whites and 2.0 times greater for blacks).

The upper limit of normal blood lead has been revised downward as new data have identified biochemical or functional changes at lower levels of blood lead. A growing body of knowledge indicates that lower levels of lead exposure than those previously recognized are expressed in altered neuropsychological function[34] and intelligence deficits.[20,34,35] Specifically, Needleman et al.[34] identified reduced general intelligence quotients (especially verbal intelligence quotients), reduced auditory or speech processing, and attention deficits among children with higher dentine lead, as compared with those who had lower dentine lead. Yule et al.,[35] in a study of 166 children whose blood lead levels ranged from 7 to 33 μg per deciliter (0.33 to 1.59 μmol per liter), reported decreases in attainment scores on tests of reading, spelling, and intelligence, but not mathematics, as blood lead levels increased. Some (but not all) of this variability was removed after the social class of the subject's family was considered. At least six prospective studies are under way in several countries to determine the extent of influence of lead exposure on the development and function of the central nervous system in children.

Heme synthesis is impaired among children with blood lead levels of <30 μg per deciliter (<1.45 μmol per liter).[36] Numerous other metabolic changes associated with low-level lead exposure have been identified. For example, in children, plasma levels of 1,25-dihydroxyvitamin D (the vitamin D metabolite that is active in stimulating gastrointestinal absorption of calcium and phosphorus) decreased as the blood lead level increased.[37] A strong negative correlation between plasma 1,25-dihydroxyvitamin D and blood lead concentrations of 12 to 120 μg per deciliter (0.58 to 5.79 μmol per liter) occurred, with no difference in the slope of the regression line for blood lead levels over or under 30 μg per deciliter (1.45 μmol per liter).[38]

Contrast with High-Risk Groups

The screening methods used and populations surveyed in the NHANES II and in the community-based lead-poisoning-prevention programs were inherently different. Therefore, estimates of the prevalence of elevated blood lead levels in children from these programs are not expected to be directly comparable. In-

stead, the NHANES II was designed to provide data on the distribution of blood lead levels for assessing the relative risks of exposure to lead in selected subgroups of the population. It is known that a large number of children with lead toxicity are included in previously identified high-risk groups. During a six-month period (October 1, 1980, to March 31, 1981) 59 programs for the prevention of childhood lead poisoning identified 10,492 cases of lead toxicity.[7] Clinical management was required in 6060 children who were at urgent and high risk.[7] In addition, individual hospitals and clinics identify many more children with lead toxicity. Although a number of cases of lead toxicity are identified through current lead-screening groups, the NHANES II data presented here indicate that large numbers of persons with elevated blood lead remain undetected, especially among preschool black children from low-income households.

The second high-risk population comprises workers occupationally exposed to lead. Baker et al. reported blood lead concentrations of ≥60 μg per deciliter (≥2.90 μmol per liter) among 67 per cent, 79 per cent, and 83 per cent of employees at three plants.[39] The small number of adult subjects (five) identified in the NHANES II with blood lead concentrations of ≥60 to <70 μg per deciliter (≥2.90 to <3.38 μmol per liter) only emphasizes the contrast between the general population and workers occupationally exposed to lead.

Estimates of the prevalence of elevated blood lead concentrations in the general population are useful information for a variety of health-assessment and planning programs.

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Biokinetic Modelling for Mammalian Lead Metabolism

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A biokinetic model of lead metabolism has been developed from data obtained in controlled single dose and chronic lead exposures of infant and juvenile baboons. The model is fitted to blood and organ clearance data after single exposures, to dynamic blood lead measurements at constant exposures, and to steady state blood lead and organ lead concentrations. The resulting model consists of a short term gut and extracellular fluid compartment, blood, liver, kidney and bone compartments. Blood lead is accurately fitted for periods of rapid change and tissue lead levels are well fitted for data from animals which were not used to develop the model.

The model has been tested for prediction of human organ burdens by comparison to a substantial body of autopsy data. The fits are satisfactory, but a respiratory compartment is desirable to complete the model.

The model is based upon chronic lead ingestion experiments in a total of sixty-six infant and juvenile baboons. Fourteen of these cases were used to establish the response of blood lead concentration to the chronic ingestion of lead acetate or lead hydroxycarbonate (one case). Whole kidney and liver and blood lead relationships were defined in 24 and 21 cases, respectively, while thirty cases were used to establish the relationship between the factors of age, lead exposure and pattern of growth with whole long bone lead concentration.

In addition, this work used data from related single exposure experiments of ^{210}Pb in the immature and adult baboon, as well as appropriate human data to derive a compartmental model of lead metabolism in the infant and juvenile baboon.

The model is based on first order kinetics. That is, a constant fraction of the substance is removed from the compartment per unit time and the time required for the compartment to initially attain a homogeneous distribution of the substance is assumed to be short relative to the biological transfer time out of the compartment.

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A series of differential equations describes the input and output for each organ. These individual organ transfer equations are then assembled into the overall equation, describing the blood lead concentration. This equation includes terms for daily input, excretion and transfers to and from each organ.

A schematic for the expected compartmental model for lead is given in Figure 1. The tissues shown contain over 90% of the total body burden of lead. The remainder is distributed at low concentrations in other soft tissues which form a large fraction of total body mass. Available data cannot support development of a more detailed model.

The expressions describing a single exposure may be readily applied to multiple exposures by modification to include an input term, P, which is the quantity per unit time going to the blood from the environment. We assume that the general expression for every compartment is:

$$Q_{x,n} = Q_{x,n-1} + dQ_{x,n}/dt \tag{1}$$

where: x = compartment x
n = day n
n-1 = day n-1

Thus, for blood we would have:

$$Q_{1,n} = Q_{1,n-1} + P + \lambda_{21}Q_2 + \lambda_{31}Q_3 + \lambda_{41}Q_4 - \lambda_{12}Q_{1,n} - \lambda_{13}Q_{1,n} - \lambda_{14}Q_{1,n} - \lambda_{15}Q_{1,n} \tag{2}$$

$$\Delta Q = Q_{1,n} - Q_{1,n-1} = P + \lambda_{21}Q_2 + \lambda_{31}Q_3 + \lambda_{41}Q_4 - \lambda_{12}Q_{1,n} - \lambda_{13}Q_{1,n} - \lambda_{14}Q_{1,n} - \lambda_{15}Q_{1,n} \tag{3}$$

The excretion of lead in bile is accounted for. Reabsorption of this lead if it occurs is accounted for in the net fractional uptake of the daily intake to the gut.

The organ contents and blood may then be readily evaluated with a simple numerical calculation suitable for computer processing. The routine calculates the incremental values, Q, for a small time step for each organ, and then tallies the current organ burden. In this way, the content of each organ is calculated on a day-by-day basis (or for a time step commensurate with the rate coefficients).

The model takes into account dynamic changes due to growth and weight change during the experimental period so that organ concentration can be determined. The body weight of each animal was measured at the beginning of the test period and then once every two weeks during the period of study. The intent of the model is